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REVIEWS

Spotlight

Climate Change and Insect Pests: Resistance Is Not Futile?

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Chemical signals produced by plants when attacked by herbivores play a crucial role in efficient plant defence. A recent study suggests that herbivore-specific R-gene resistance may be enhanced by elevated atmospheric CO_2 concentrations. Understanding how climate change affects plant resistance to herbivorous pests could be essential for future food security.

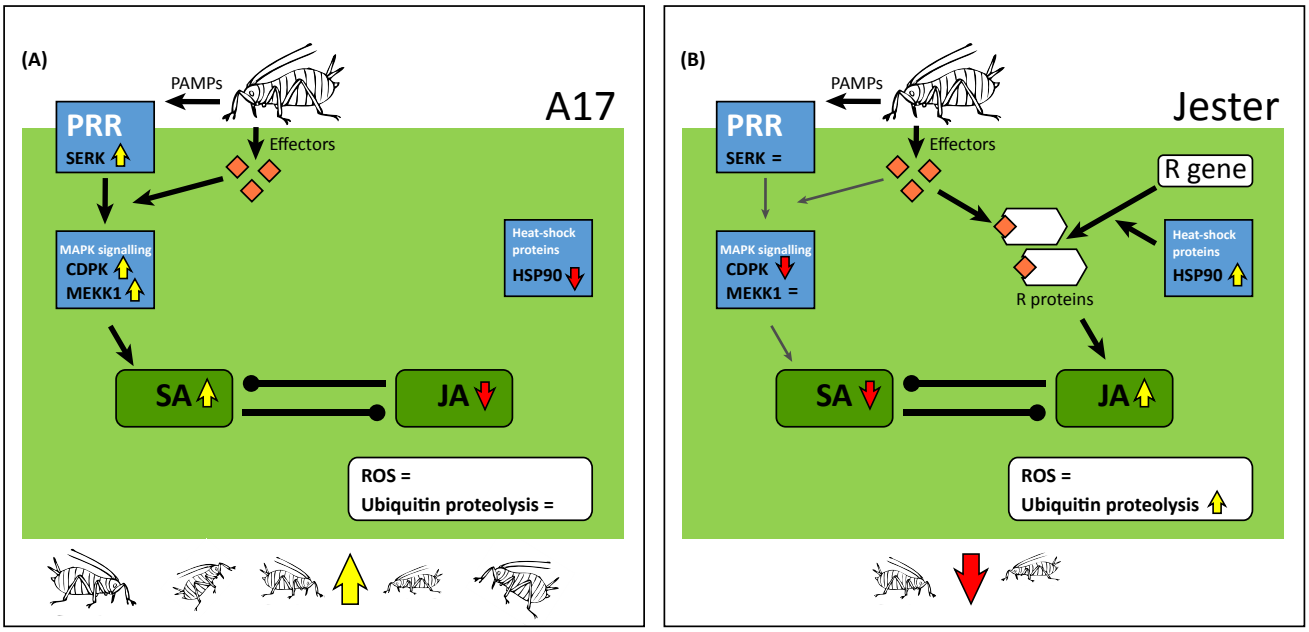
With human populations expected to reach 11.2 billion by 2100, global food security has become an urgent priority. Insect pests currently reduce crop productivity worldwide by an average of 14% [1]. As many pests are anticipated to become more problematic with climate change (we include increases in atmospheric carbon dioxide in this) [2], achieving global food security must involve effective crop protection. Aphids already cause significant damage to many crop plants at a global scale [3], and comparative meta-analyses (e.g. [4]) usually show that aphids benefit from elevated concentrations of carbon dioxide (e[CO_2]) compared to other herbivorous groups (though not universally). A recent study by Sun *et al.* [5], however, reported that some plants may resist aphids much better under e[CO_2] because of increased resistance (R) gene activity.

Plant Resistance to Herbivores – a Co-evolutionary Arms Race

Plants and herbivorous insects have been locked in a co-evolutionary arms race for 350 million years. Plants deploy an arsenal of physical and chemical defences against herbivorous insects, to which herbivores evolve counter-adaptations. In turn, plants may themselves evolve adaptations to cope with adapted herbivores. Within the last decade, common frameworks have emerged for investigating such plant–insect interactions [6] (Figure 1). Plants generally perceive attackers using pattern recognition receptors (PRRs) upon contact with conserved patterns of molecules associated with the attacker; microbial/pathogen/herbivore – associated molecular patterns (MAMPs, PAMPs, HAMPs respectively) [7] {for consistency with Sun *et al.* [5] we adopt (P)athogen versions of the acronyms from hereon}. This leads to PAMP-triggered immunity (PTI) involving activation of downstream phytohormone-dependant signalling pathways as key regulators of plant resistance to arthropod herbivores: the jasmonic acid (JA) pathway activates resistance against herbivores (especially chewing herbivores), while the salicylic acid (SA) pathway is involved in resistance against pathogens [8]. Many adapted herbivores produce effectors (e.g. orally secreted proteins) that modulate plant defensive pathways and may suppress or manipulate PTI [7]. Aphids are particularly successful in this regard, often producing multiple effectors in their saliva [9]. Because of their unique mode of feeding, aphids mostly trigger SA-mediated responses in their host plant, even though JA-mediated defences would often be more effective against them [3]. Aphids may thus actively induce the SA pathway to suppress JA-mediated defences via phytohormonal crosstalk (Figure 1A). However, it should be noted that SA-mediated defences provide potent aphid resistance in some plants, thus an upregulation of the SA pathway may not be universally beneficial for the aphid [3]. In response to the aphid's manipulation of plant defences with effectors, some plant biotypes [possessing R (resistance) genes] can produce R proteins that recognise insect effectors and activate effective downstream defence pathways, leading to effector triggered immunity (ETI, Figure 1 B) [7]. In essence, PTI is based on non-specific recognition of common PAMPs and can be suppressed or hijacked by aphid effectors, whereas ETI is prompted by the recognition of specific compounds associated with the herbivore's attempts to manipulate plant defences [7,8].

Plant Resistance and Climate Change

Significantly, these specific resistance mechanisms have not been widely studied in the context of climate change, even though they may contribute to existing crop resistance to insect pests. Breakdown of such resistance could therefore leave crops more susceptible to herbivores, impacting productivity and potentially food security [2]. The study of Sun *et al.* [5] is significant because it is, to our knowledge, the first to provide a mechanism by which e[CO_2] could affect ETI and PTI defences. The study compared resistance of *Medicago truncatula* to the pea aphid (*Acyrthosiphon pisum*) using two plant genotypes with different resistance strategies. One (cv. Jester) possesses an R-gene which confers resistance to some Australian *A. pisum* clones, and therefore Jester predominantly expresses ETI resistance; the other (A17) does not possess the R-gene and should rely primarily on PTI resistance [5]. Environmental stress (e.g. drought, salinity and temperature) can affect R gene resistance, with two studies in arabidopsis (*Arabidopsis thaliana*) linking this to reduced R protein activity and downstream signal transduction (see [5] for references). Central to this is the fact that R protein structure and the initiation of signal transduction of ETI defences relies on a specific subclass of heat-shock



Trends in Plant Science

Figure 1. Simplified Depiction of Defensive Responses of Plants Challenged by Herbivores Involving Various Components of the Signalling Pathway. (A) PAMP (Pathogen-Associated Molecular Pattern)-triggered immunity (PTI): PAMPs (and some herbivore-associated molecular patterns, HAMPs) are recognised by the plant's Pattern Recognition Receptors (PRR), which initiate mitogen-activated kinases (MAPK) signalling cascades that result in the upregulation of salicylic acid (SA)-mediated defence. Aphids may inject effectors into the plant to suppress PTI where they are susceptible to SA-mediated defences. Conversely, where they are more susceptible to jasmonic acid (JA)-mediated defences, aphids may use effectors to actively stimulate PTI, thereby suppressing JA via phytohormonal crosstalk (blunt arrows). Upward (yellow) and downward (red) arrows indicate increased and decreased activity in response to e[CO_2] as reported by Sun *et al.* [5]. The = symbol indicates no change in response to e[CO_2]. In A17 plants, e[CO_2] increased PRR and plant kinase expression (somatic embryogenesis receptor-like kinase, SERK; calmodulin-like domain protein kinase, CDPK; mitogen-activated protein kinase kinase kinase 1, MEKK1), resulting in upregulation of the SA pathway and suppression of the JA-pathway to the aphid's benefit. (B) Effector-triggered immunity (ETI): Plants with (resistance) R-gene-based resistance produce R proteins which are capable of interfering with and recognising aphid effectors and upon recognition, activate a strong JA-mediated defence response. A class of heat shock proteins (cytosolic heat shock protein 90, HSP90, and its co-chaperones) can stabilize R proteins, which increases efficiency of R-gene mediated defence. In Jester plants, e[CO_2] enhanced production of heat shock proteins, which facilitated R protein activity against effectors and upregulation the JA-pathway and ubiquitin proteolysis, resulting in reduced aphid abundance. Reactive oxygen species (ROS), another trait commonly associated with herbivore resistance, were induced by aphid feeding on both plant genotypes, but were unaffected by e[CO_2].

protein (HSP90). While HSP90 responds negatively to the abiotic factors above, it can be upregulated under e[CO_2] [10]. The overarching hypothesis was therefore that e[CO_2] could promote HSP90 gene expression in *M. truncatula*, resulting in increased R gene stability and R gene-dependent defences (i.e. ETI) against *A. pisum* [5].

The abundance of *A. pisum* was significantly higher on A17 plants (R gene absent) when grown under e[CO_2] compared to ambient CO_2 (a[CO_2]). Simultaneously, several marker genes of PTI resistance were upregulated in response to e[CO_2], including the PRR gene somatic embryogenesis receptor-like kinase (SERK) and downstream mitogen-activated kinases (MAPKs), resulting in an activation of SA signalling pathways, but decreases in JA activity (Figure 1A). This suggests that *A. pisum* actively stimulates PTI resistance of A17 plants to suppress a more potent JA response of the plant. In direct contrast, e[CO_2] decreased performance of *A. pisum* on Jester plants (R gene present) compared to aphids grown at a[CO_2] (Figure 1B). Increased aphid resistance was linked to the upregulation of heat shock proteins (HSP90) and co-chaperones (RAR1, SGT1), which increased ETI efficiency and strongly activated downstream plant responses including JA signalling and ubiquitin-mediated proteolysis. Further support for the importance of heat shock proteins was provided by the silencing of the HSP90 gene in Jester plants; JA activity and ubiquitin-mediated proteolysis were impaired and the enhanced resistance to aphids observed under e[CO_2] was negated [5].

Studies exploring the effect of e[CO_2] on R gene resistance to herbivores are rare and have so far reported variable responses. For example, *Rubus idaeus* cultivars

possessing the A_1 R gene became more susceptible to the European large raspberry aphid (*Amphorophora idaei*) under e[CO_2], whereas resistance to *A. idaei* was unchanged for cultivars possessing the A_{10} R gene [11]. In a case analogous to R-gene resistance, e[CO_2] caused down-regulation of genes associated with defence signalling hormones ethylene (acc) and JA (lox 7, lox 8) in soybean (*Glycine max*) cultivars possessing cysteine proteinase inhibitors (CystPI, which interfere with digestive functions in many coleopterans). This resulted in reduced production of CystPI and enhanced performance of the Japanese beetle (*Popillia japonica*) and western corn rootworm (*Diabrotica virgifera virgifera*) [12].

Concluding Remarks

R gene resistance is highly specific and can be limited to a single clone of an insect species [7]. Sun *et al.* [5] readily acknowledge this limitation and highlight that R genes are ineffective against some European clones of *A. pisum*. Indeed, resistance based on single genes is under strong selective pressure and herbivores – particularly aphids, which have formidable generation rates – can frequently overcome monogenic resistance [3]. ETI defence, for example, can be overcome by aphids through diversification, modification, or loss of the effector gene, or by deploying novel effectors to suppress defences [7]. Predicting the wider significance and consequences of e[CO_2] on plant resistance, particularly against aphids, is therefore a risky endeavour. Nonetheless, we regard the study by Sun *et al.* [5] to represent an important step forward in addressing how climate change might affect plant–herbivore interactions. In particular, this study presents a mechanistic explanation for how e[CO_2] is affecting ETI and PTI defences at a molecular level and directly relate this to the performance and feeding behaviour of the model herbivore.

We are now at the stage where we should aim to relate herbivore responses to e[CO_2] more directly to generic plant defence signalling pathways that are common across plant taxa, such as the PTI/ETI model [6]. While the PTI/ETI model is an oversimplification, it has proved useful for making conclusions about the specificity of plant recognition of its antagonists and consequent deployment of plant defences [8]. Aphids, in particular, could be a good model for investigating the effects of e[CO_2] on ETI and PTI defences against herbivores an increasing number of R genes and effector proteins are being identified in this plant–herbivore system.

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References

1. Ferry, N. and Gatehouse, A.M.R. (2010) Transgenic crop plants for resistance to biotic stress. In *Transgenic Crop Plants* (Kole, C. *et al.*, eds), pp. 1–65, Springer-Verlag
2. Gregory, P.J. *et al.* (2009) Integrating pests and pathogens into the climate change/food security debate. *J Exp. Bot.* 60, 2827–2838
3. Züst, T. and Agrawal, A.A. (2016) Mechanisms and evolution of plant resistance to aphids. *Nat. Plants* 2, 15206
4. Robinson, E.A. *et al.* (2012) A meta-analytical review of the effects of elevated CO_2 on plant–arthropod interactions highlights the importance of interacting environmental and biological variables. *New Phytol.* 194, 321–336
5. Sun, Y. *et al.* (2018) Elevated CO_2 increases R gene-dependent resistance of *Medicago truncatula* against the pea aphid by up-regulating a heat shock gene. *New Phytol.* 217, 1696–1711
6. Wu, J.Q. and Baldwin, I.T. (2010) New insights into plant responses to the attack from insect herbivores. *Annu. Rev. Genet.* 44, 1–24
7. Hogenhout, S.A. and Bos, J.I.B. (2011) Effector proteins that modulate plant-insect interactions. *Curr. Opin. Plant Biol.* 14, 422–428
8. Erb, M. *et al.* (2012) Role of phytohormones in insect-specific plant reactions. *Trends Plant Sci.* 17, 250–259
9. Will, T. *et al.* (2013) How phloem-feeding insects face the challenge of phloem-located defenses. *Front. Plant Sci.* 4, 336
10. Li, P.H. *et al.* (2008) Arabidopsis transcript and metabolite profiles: ecotype-specific responses to open-air elevated [CO_2]. *Plant Cell Environ.* 31, 1673–1687
11. Martin, P. and Johnson, S.N. (2011) Evidence that elevated CO_2 reduces resistance to the European large raspberry aphid in some raspberry cultivars. *J. Appl. Entomol.* 135, 237–240
12. Zavala, J.A. *et al.* (2008) Anthropogenic increase in carbon dioxide compromises plant defense against invasive insects. *Proc. Natl. Acad. Sci. U. S. A.* 105, 5129–5133

Spotlight

Ligands Switch Model for Pollen-Tube Integrity and Burst

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In flowering plants, pollen tubes deliver and release the immotile sperms to the female gametes for fertilization, but mechanisms remain unclear. New results show that the RALF family peptides control pollen-tube integrity and burst by binding to CrRLK1L family receptors and cell wall leucine-rich repeat extensins.

During the adaptive evolution of flowering plants, sperm lost its mobility and a pollen tube, which is germinated from the male gametophyte (pollen), was evolved to deliver the sperms to the female gametophyte (embryo sac). The structure of gametophytes is largely reduced, with a seven-celled embryo sac and a three-celled pollen grain. The embryo sac typically contains two synergids, one egg, one central cell, and three antipodal cells. The synergid is crucial for pollen-tube attraction and reception, as well as pollen-tube burst to release the sperm cells. These processes require molecular signaling between the pollen tube and the synergid [1].

On the female side, multiple components crucial for pollen-tube reception in the synergid have been reported. FERONIA (FER), a member of the CrRLK1L subfamily, regulates the membrane targeting of